Australia Antigen and Viral Hepatitis

The discovery of Australia antigen in the 1960's has provided a valuable marker for epidemiologic studies of viral hepatitis. Initial reports linked Australia antigen with both "infectious" and "serum" hepatitis, however these studies were based on sporadic hospitalized cases of hepatitis in whom the sources of infection often were poorly defined. Subsequent experimental studies have firmly established the specific association of Australian antigen with serum hepatitis. In addition, there have been numerous reports from throughout the world documenting the failure to detect Australia antigen in common-source outbreaks that were clinically and epidemiologically typical of infectious hepatitis.

Until recently, the prevailing view has been that serum hepatitis was transmitted exclusively via the parenteral route and was not a contagious disease. This concept has been modified as studies have shown that Australia antigen-associated serum hepatitis can be transmitted orally as well as parenterally and can spread to contacts, although rather less regularly than infectious hepatitis. These studies confirm isolated epidemiologic observations which suggested that serum hepatitis could be transmitted by close contact as well as by parenteral exposure. Australia antigen has been demonstrated in urine as well as serum. Though postulated, the presence of antigen in feces has not been demonstrated unequivocally at the present time. The observations that both forms of viral hepatitis can be transmitted by the oral and parenteral routes have added force to the non-committal terms type A and type B hepatitis to describe "infectious" short-incubation and "serum" long-incubation forms of the disease, respectively. Type B hepatitis is transmitted more readily by exposure to blood or blood products, because, as indicated by the presence of Australia antigen, the virus may persist for a long time in some patients, particularly those with anicteric disease who become "healthy" carriers.

The demonstration that type B hepatitis can be spread by contact exposure raises an interesting question regarding the marked increase of viral hepatitis in California during the last several years. The increased incidence has been associated with a marked change in the age distribution of cases. Up to 1960, peak age-specific incidence occurred in children less than 14 years of age, an age pattern epidemiologically consistent with the predominance of type A hepatitis. Since 1960 the incidence in those less than 14 years has remained essentially unchanged, while the incidence in persons 15 to 29 years of age has skyrocketed to rates five times greater than those in children. About twenty percent of the cases in young adults are reported as associated with illicit parenteral drug use. However, no such association is made in the remaining 80 percent.

Coincident with the increase of viral hepatitis in young adults have been profound changes in lifestyle among segments of this age group. Drug use has become widespread, frequently coupled with crowded living conditions, poor attention to personal hygiene and substandard sanitation. Although a matter of speculation at this time, it may be that a significant proportion of viral hepatitis in young adult Californians is in fact contact-acquired, Australia antigen-associated, type B hepatitis. The true extent of the type B hepatitis "iceberg" in adults should become more evident as Australia antigen testing becomes an integral part of the viral hepatitis diagnostic workup. Physicians are urged to include Australia antigen test results when reporting patients with this disease to the health department.

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REFERENCES

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